Email from Dr. Pamela Mink: 02 28 2006 03:42 PM

Dear Mr. Miller,

I am attaching a Word document that contains the comments I presented on today's arsenic SAB panel call. I appreciate your distributing this as soon as possible to the Panel members, given that several people mentioned that they couldn't hear my verbal comments due to problems with the phone connection.

Thanks very much,

Pam Mink

<<Mink comments for Feb 28 call.doc>>

Pamela Mink, Ph.D., M.P.H.

Managing Scientist

Health Sciences

Exponent, Inc.

1730 Rhode Island Ave., NW

Suite 1100

Washington DC 20036

Phone: 202-772-4921 Fax: 202-772-4979

Thank you for the time to address the Panel today.

My name is Pamela Mink and I am an epidemiologist and managing scientist with Exponent in Washington, DC. I am speaking at the request of the Wood Preservative Science Council.

My comments today are based on the revised draft and written comments by panel members, specifically as they relate to Question C2. This was also one of the topics covered on today's call.

Many recommendations by panel members address important points which I urge the committee to incorporate into the final draft of their report.

First, the limitations of the SW Taiwan dataset must be considered fully, and further analyses as recommended by some Panel members, including sensitivity analyses and analyses to evaluate the validity of the study, are clearly warranted. Even though published studies based on these data have undergone some degree of peer review, to my knowledge, there has been no formal quantitative evaluation of validity; or in other words, of the impact of bias. Furthermore, I agree with the comments noted in the text of the draft that the Taiwan dataset alone is not sufficient for estimating cancer risk in humans. Therefore, it is necessary to turn to additional epidemiologic studies and to integrate these into the analysis.

Second, it should be recognized that the epidemiologic case-control and cohort studies have the advantage of the ability to assess individual data on exposure. In the study by Steinmaus et al. (2003), the investigators based assessment of arsenic exposure on drinking water arsenic concentrations, volume of water consumption, use of bottled water, and the use of water filters that remove arsenic. These authors also analyzed their data according to both exposure level and latency, as well as by exposure level at several intervals ranging from 10-50 years prior to diagnosis. As another example, the study by Bates et al. (1995), conducted in Utah, restricted the main analyses to cases and controls who had lived in study towns for at least half of their lives prior to the diagnosis of bladder cancer. This addresses concerns about residential stability of study participants. I mention these studies to illustrate that, while any observational study will have limitations, it is also possible to conduct thorough analyses that take into account individual differences in water consumption behavior, changing exposures over time, and timing of exposure with respect to disease onset. Time constraints prevent me from discussing additional studies in detail; however analyses such as those I just described are not possible in ecologic studies, where group-level rather than individual-level data are collected and analyzed.

Finally, with respect to the sections inserted on page 32 of the draft document, it is both appropriate and feasible to integrate data from multiple epidemiologic studies. It is important to include this section, so that EPA can review and evaluate these methods as they consider options for conducting a risk analysis that may incorporate data from more than one study.

Again, as I have urged the committee previously, an integrative analysis can be used not only to assess "concordance" with a primary model, but the results of such an analysis can also provide the basis of a primary model, as has been done in the EPA risk assessment for methylmercury.

Thank you again for your time.